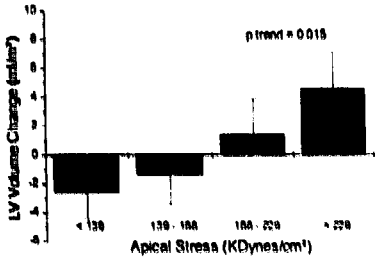
Methods: Utilizing finite element methods, we tested whether increased wall stress post MI predicts LV remodeling. 64 patients with acute anteroseptal MI and high quality echo images were selected from the Healing and Early Afterload Reducing Therapy Trial, a trial of ACE inhibition following anterior MI. 31 patients received low dose (0.625 mg) ramipril and 33 received full dose (10 mg) ramipril. End-systolic LV models were constructed from orthogonal apical views obtained by echo two weeks post MI. LV wall stress, assessed by finite element methods, was compared with subsequent change in LV volume (14–90 days).

Results: LV volume change increased across quartiles of apical wall stress (p, trend = 0.015) in all patients, and in the low dose group (p, trend = 0.004), but was attenuated in the full dose group (p, trend = 0.72). Apical wall stress remained highly predictive of volume change after adjusting for infarct size and heart size in all patients (r = 0.58; p = 0.03) and in the low dose group (r = 0.71; p = 0.004).



Conclusions: Apical LV wall stress 2 weeks post MI predicts subsequent LV remodeling, and this relationship is attenuated by full dose ACE-inhibition, suggesting that ACE inhibition may modify the relationship between regional LV wall stress and remodeling.

2:15

894-2 Sympathetic Denervation in Patients With Chronic Coronary Artery Disease: Relationship to Myocardial Perfusion and Metabolism

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Cardiac sympathetic denervation has been previously described in pts with coronary artery disease (CAD) following acute myocardial infarction, but there are little data correlating sympathetic innervation with perfusion and metabolism in patients with chronic coronary artery disease and ventricular dysfunction. PET imaging of myocardial perfusion (with nitrogen-13 ammonia, NH3), metabolism (with fluorine-18 fluorodeoxyglucose, FDG) and sympathetic innervation (with carbon-11 hydroxyephedrine, HED) was performed in 8 nondiabetic men, 54 ± 15 yrs, with known CAD referred for evaluation of myocardial ischemia and viability. A semi-automated program was used to determine NH3 and FDG uptake and HED retention in 177 regions of interest encompassing the left ventricular (LV) myocardium. Perfusion, metabolism and innervation defects were defined as the percentage of LV with tracer uptake or retention >2 SD below values obtained from a normal database. The extent of defects with each tracer was as follows (mean ± SD, range): NH3 37.3 ± 13.2% (21.7–61.1), FDG 44.2 ± 19% (19.9–75.6), and HED 36.5 ± 11.4 (18.1–51.8), p = NS for tracer effect.

Conclusions: In those patients with chronic CAD and ventricular dysfunction, the extent of sympathetic denervation matches the extent of ischemic injury and reduced glucose metabolism. Since the extent of sympathetic denervation exceeds the extent of ischemic injury acutely after MI, these results could be explained by sympathetic reinnervation during the recovery period.

2:30

894-3 Comparison of Dobutamine Stress Tagged MRI With F-18 FDG PET to Assess Myocardial Viability in Patients With Chronic Ischemic Heart Disease

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Background: Characterizing the jeopardized but salvageable myocardium represents a key step in patient management after myocardial infaction. We evaluated regional strains in patients with chronic ischemic heart disease and compared myocardial viability assessed by strain measurements to viability assessed using FDG PET.

Materials and Methods: 14 patients with a history of myocardial infarction were referred to assess myocardial viability. Tagged breath-hold MRI (6 slices) was acquired at rest, and under low-dose dobutamine infusion (10 μg/kg/mn). Strain maps at rest and under stress were computed. FDG-PET images were acquired in 3D mode with a CTI/HR+ camera. Based on coronary angiography, clinical history and PET imaging, regions were assigned to one of the following groups: infarcted and non-viable regions (glucose uptake <50%), infarcted but viable regions (glucose uptake ≥70%), non-infarctod but ischemic regions, and normal regions.

Results: End-systolic maximum shortening (MS) remained severely depressed under stress (−1 ± 6%) in infarcted regions, whereas angular deviation ranged from −30° to 50°. When circumferential extent of infarcted regions with a glucose uptake <50% was of 2 or more segments, we observed larger end-systolic MS associated with large angular deviation that was corresponding to radial stretching. Maximum shortening was significantly larger (p < 0.05) in all viable regions but remained inferior to normal regions (MS: −17 ± 3%, angle: −13 ± 6°)

Conclusion: Tagged MR imaging and strain maps can differentiate nonviable and viable regions in patients with chronic ischemic heart disease.

2:45

894-4 Differential Effects on Left Atrial Function of Pacing-Induced Ischemia in Patients With Diseased Proximal Left Anterior Descending and Proximal Left Circumflex Coronary Artery

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In left ventricular (LV) ischemia, compensatory augmentation of left atrial (LA) contraction enhances LV filling and performance, whereas loss of this atrial transport function exacerbates hemodynamic compromise. We hypothesized that one mechanism for the loss of this important enhancer of LV performance may be ischemic LA dysfunction. LA function was therefore compared in 8 patients with single-vessel left anterior descending (LAD, group A) and in 8 patients with single-vessel proximal left circumflex (LCx, group B) coronary artery stenosis at rest and immediately after pacing. LV and LA pressure-area relations were derived from simultaneous double-tip micromanometer pressure recordings and automatic boundary detection echocardiograms. Immediately after pacing, LV end-diastolic pressure rose from 8.8 to 18.7 mmHg in group A and from 9.4 to 19.5 mmHg in group B (p < 0.001 vs rest for both groups), time constant of relaxation increased from 33.9 to 41.2 msec in group A (p < 0.01 vs at rest) and from 34.2 to 55.6 mmHg in group B (p < 0.001 vs rest), LA mean pressure rose from 9.3 to 19.2 mmHg in group A and from 10.4 to 19.2 mmHg in group B (p < 0.001 vs rest for both groups) and LV stiffness increased from 0.098 to 0.202 cm^{-2} in group A and from 0.096 to 0.194 mmHg in group B (p < 0.001 vs rest for both groups). All these changes were similar in both groups. However, LV peak systolic pressure fell from 127.7 to 111.9 mmHg in group B while it remained unchanged in group A, LA systolic emptying index decreased from 0.26 to 0.15 in group B while it increased from 0.28 to 0.32 in group A (p < 0.001 vs rest for both groups), LV stroke work index decreased from 1053.4 to 749.7 $mmHg.cm^2$ in group B (p < 0.05) without a significant change in group A, the area of the A loop of the LA pressure-area relation decreased from 14.4 to 9.3 $mmHg.cm^2$ in group B (p < 0.05 vs rest) while it increased from 12.4 to 54.1 $mmHg.cm^2$ in group A (p < 0.01 vs rest) and LA stiffness increased from 0.200 to 0.287 in group B (p < 0.001 vs rest) without a significant change in group A.

Conclusions: In patients with LAD coronary artery stenosis, LV ischemia is associated with enhanced LA function, manifest as augmented LA A loops. However, in patients with proximal LCx coronary artery stenosis, LA branches might have been affected, rendering the LA ischemic and unable to increase its booster pump function. Ischemic depression of LA function contributes to further decrease of LV performance.

3:00

894-5 Anterior Myocardial Ischemia Causes Simultaneous Left and Right Ventricular Contractile Dysfunction

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The contribution of the interventricular septum to global RV contractile function remains unknown. In order to examine the effects of septal and LV free wall ischaemia on LV and RV contractile performance, 8 open-chest, open pericardium pigs underwent mid LAD occlusion for 50 mins followed by 60 mins of reperfusion. A 6F integrated conductance catheter and micromanometer was inserted into both the LV and RV to assess simultaneous pressure and volume changes at baseline, during LAD occlusion, and at 15 and 60 mins of reperfusion. Ventricular volumes were calibrated from a transit time flow probe placed around the pulmonary artery and contractile indices were obtained from pressure-volume relations (PVR) during IVC occlusion.